62 EPITOMES—NUCLEAR MEDICINE

preparation. Excellent correlation with radionuclide cerebral angiography has been shown. This is likely to become the technique of choice for confirming brain death.

C. L. LUTRIN, MB, ChB Sacramento, California

REFERENCES

Laurin NR, Driedger AA, Hurwitz GA, et al: Cerebral perfusion imaging with technetium 99m HM-PAO in brain death and severe central nervous system injury. J Nucl Med 1989; 30:1627-1635

Report of the Medical Consultants on the Diagnosis of Death to the President's Commission for the Study of Ethical Problems in Medicine and Biomedical and Behavioral Research: Guidelines for the determination of death. JAMA 1981; 246:2184-2186

Spieth ME, Ansari AN, Kawada TK, Siegel ME: Comparison of DTPA and HMPAO for the evaluation of brain death. J Nucl Med 1991; 32:1839

Radionuclide Therapy for Thyroid Disease

ONE OF THE GOALS OF therapy is to deliver the active agent directly to the disease site and to have the rest of the body subjected to as little of the agent as possible. This principle is even more important when therapy with systemic radionuclides is considered. Iodine therapy for thyroid diseases meets this goal.

Radioiodine has a role as a primary treatment of hyperthyroidism. It also has a role in the treatment of differentiated papillary and follicular thyroid cancer. Of the hyperthyroid states, the most common is Graves' hyperthyroidism. This is ideally treated with radioiodine because the gland is diffusely involved and the uptake of radioiodine is usually greater than 50% of an administered dose. Patients of all ages can be treated. Contraindications to treatment are pregnancy and breast feeding.

Although over the years there has been debate about the appropriate therapeutic dose, some investigators try to select a dose that would bring the patient to a euthyroid state. Long-term experience, however, has dictated that this goal is often not achievable. I now advise prescribing one therapeutic dose designed to cure hyperthyroidism with the expectation that permanent hypothyroidism will occur. The patient should be counseled and recognize the need for life-long thyroid hormone replacement.

Single toxic nodules and toxic multinodular goiters can also be treated with radioiodine therapy. These conditions, in general, are somewhat more resistant to irradiation, and the prescribed dose is proportionately greater. In contrast to Graves' disease where the thyroid becomes impalpable after the administration of sodium iodide I 131, nodular goiters treated with radioiodine often are still palpable and the patient is rendered euthyroid. The incidence of posttreatment hypothyroidism is lower after the treatment of nodular goiters with ¹³¹I-sodium iodide. Long-term follow-up has failed to show an increased risk of cancer or genetic abnormalities in the offspring of patients treated with ¹³¹I-sodium iodide.

Radioiodine therapy for thyroid cancer is an adjuvant treatment that is given in selected patients after the primary lesion has been removed surgically. It is frequently used to ablate remnants of presumed normal thyroid after the operation. The exact role of this is still open to debate. It can also be used to treat functioning metastases in regional lymph nodes and in distant organs such as the lungs or bones. A preparatory whole-body scintiscan using ¹³¹I shows the extent of disease. Should this treatment be considered, it is extremely important to discontinue exogenous thyroxine therapy and to demonstrate that the thyrotropin level is elevated. It is also important to ensure that large doses of exogenous iodine, in particular, radiographic contrast, are not given for

several weeks before radioiodine therapy. If a dose of 30 mCi or more is prescribed, the patient should be admitted to hospital until the retained dose falls below that level. This treatment is well tolerated compared with systemic chemotherapy and external beam therapy. The long-term hazards that might be anticipated, including the occurrence of second malignant neoplasms, have not been described in several large followup studies.

Serious complications of radioiodine therapy such as thyroid storm and acute thyroiditis are extremely uncommon. Data published recently do not support a relationship between the occurrence of infiltrative ophthalmopathy and treatment with radioiodine.

I. ROSS McDOUGALL, MB, ChB, PhD Stanford, California

REFERENCES

Cooper DS: Treatment of thyroidoxitosis, In Braverman LE, Utiger RD (Eds): Werner and Ingbar's The Thyroid: A Fundamental and Clinical Text, 7th Ed. Philadelphia, Pa, JB Lippincott, 1992, pp 887-916

Edmonds CJ, Smith T: The long-term hazards of the treatment of thyroid cancer with radioiodine. Br J Radiol 1986; 59:45-51

Hennemann G, Krenning EP, Sankaranarayanan K: Place of radioactive iodine in the treatment of thyrotoxicosis. Lancet 1986; 1:1369-1372

McDougall IR: Thyroid Disease in Clinical Practice. London, Oxford University Press, 1992

Diagnostic and Therapeutic Uses of Metaiodobenzylquanidine

METAIODOBENZYLGUANIDINE (MIBG), a guanethidine analogue developed as an adrenal imaging agent, shares a specific uptake and storage mechanism with norepinephrine. Since its introduction as a radiopharmaceutical, numerous studies have documented its high sensitivity (90%) and specificity (100%) for detecting pheochromocytoma and neuroblastoma. Consequently, MIBG has gained prominence not only in the diagnosis but also in the staging and posttherapeutic evaluation of these tumors. This agent also localizes in carcinoid (40%) and various other neuroendocrine tumors, but with less sensitivity.

When radiolabeled with either iodine 123 or iodine 131, MIBG normally localizes in the salivary glands, heart, liver, and urinary bladder with occasional uptake in lacrimal glands, normal adrenal glands, and colon. Because bone uptake is not expected in the absence of tumor involvement, osseous metastases can be easily identified.

There are several instances in which the normal biodistribution of MIBG is altered. The use of interfering medications such as tricyclic antidepressants, phenothiazine, labetalol, decongestants, and cocaine may result in a nondiagnostic or falsely negative study. These medications should be discontinued at least seven days before obtaining an MIBG scan.

Metaiodobenzylguanidine scans are indicated in several conditions. If a mass is present, an MIBG scan can indicate whether it is of neuroectodermal origin. This can be useful in hypertensive adults with incidentally discovered adrenal masses on computed tomography scans. Also, because 10% to 30% of pheochromocytomas are multiple, or extra-adrenal, these can be detected, ensuring a good surgical result. Finally, because of the occasional histologic confusion over "small blue cell" neoplasms in children, MIBG scanning can be crucial in making a diagnosis of neuroblastoma.

Compounds comprising MIBG labeled with ¹³¹I and, more recently, ¹²⁵I are under investigation as parenteral radiotherapeutic agents to be used in advanced cases of both pheochromocytoma and neuroblastoma. Initial results worldwide in about 100 patients indicate a 30% partial response rate of fair durability and a few complete responses. This is of particular importance in neuroblastoma, one of the most common solid tumors of infancy and childhood, in which widespread disease has a dismal long-term prognosis. In fact, ¹²⁵I- and ¹³¹I-MIBG offer one of the few new therapeutic strategies available for this lethal cancer.

Although the availability of MIBG has been limited in the United States, there are many institutions currently doing diagnostic studies. Centers involved in the evaluation of MIBG as a systemic radiotherapeutic agent are less numerous. The approval of MIBG by the US Food and Drug Administration, anticipated sometime this year, should increase

availability. In the interim, in view of the diagnostic benefits afforded, clinicians should not hesitate to request MIBG scans and refer to another center, if necessary.

MARGUERITE T. PARISI, MD Los Angeles, California

ROBERT S. HATTNER, MD San Francisco, California

REFERENCES

Parisi MT, Sandler ED, Hattner RS: The biodistribution of metaiodobenzylguanidine. Semin Nucl Med 1992; 22:46-48

Treuner J, Klingebiel T, Bruchelt G, Feine U, Niethammer D: Treatment of neuroblastoma with metaiodobenzylguanidine: Results and side effects. Med Pediatr Oncol 1987: 15:199-202

Von Moll L, McEwan AJ, Shapiro B, et al: Iodine-131 MIBG scintigraphy of neuroendocrine tumors other than pheochromocytoma and neuroblastoma. J Nucl Med 1987; 28:979-988

ADVISORY PANEL TO THE SECTION ON NUCLEAR MEDICINE

DAVID C. PRICE, MD
Advisory Panel Chair
CMA Council on Scientific Affairs Representative
Section Editor
University of California, San Francisco

JOHN H. MILLER, MD CMA Section Chair Los Angeles

DAVID L. LILIEN, MD CMA Section Secretary Newport Beach

DANIEL A. NAVARRO, MD
CMA Section Assistant Secretary
Oakland

ROBERT F. CARRETTA, MD Immediate Past Panel Chair Fair Oaks

JOSEP G. LLAURADO, MD Loma Linda University

I. Ross McDougall, MB, ChB, PhD Stanford University

CAL LUTRIN, MD

University of California, Davis

KENNETH LYONS, MD University of California, Irvine

RANDALL A. HAWKINS, MD University of California, Los Angeles

WILLIAM L. ASHBURN, MD University of California, San Diego MICHAEL E. SIEGEL, MD University of Southern California

RICHARD W. MYERS, MD Society of Nuclear Medicine Sacramento

HIRSCH HANDMAKER, MD American College of Nuclear Physicians San Francisco